Retraction

TAF_{II}s Mediate Activation of Transcription in the *Drosophila* Embryo

We previously reported that transcriptional activation of *hunchback* (*hb*) and *huckebein* (*hkb*) by Bicoid in the *Drosophila* embryo is impaired by mutations in $TAF_{II}110$ and $TAF_{II}60$. This conclusion was based on in situ staining of wild-type versus mutant embryos (Figures 5 and 6 of Sauer et al., Cell *87[7]*, 1271–1284, 1996). However, we have come to realize that, due to technical problems with our previous embryo staining procedures, the in vivo results in Figures 5 and 6 are incorrect and those in Figures 7–9 are uncertain. Recent data by J. Zhou and R. T. indicate that mutations in $TAF_{II}60$ (Figure 1 below) and $TAF_{II}110$ (data not shown) have no detectable effect on *hb* expression. However, the isolation and molecular characterization of these TAF_{II} mutants (Figure 1–3, Sauer et al., 1996) is correct and recent studies (see Zhou et al., 1998) suggest that these TAF_{II} mutations affect Dorsal-mediated activation of *snail* and *twist* in the *Drosophila* embryo. We thank Gary Struhl, Claude Desplan, and Mike Levine for alerting us to problems in our previous embryo staining experiments and Jumin Zhou for the results in Figure 1.

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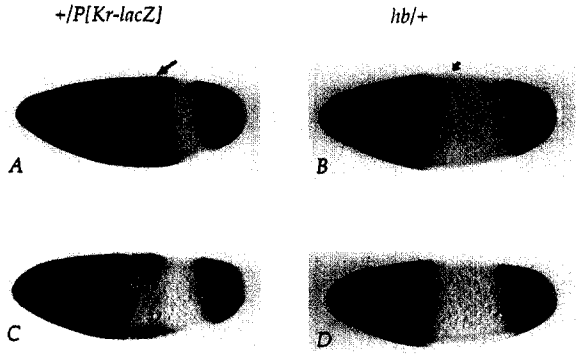

Figure 1. *hunchback* (*hb*) Expression in $TAF_{II}60^{YY}$ Mutant Embryos

Stage 5 embryos were collected from the mating of hb^{14}/*P[Kr-lacZ]* heterozygous males and either wild-type (A and B) or $TAF_{II}60^{YY}$/+ heterozygous (C and D) females. The embryos (2.5 hr postfertilization) were hybridized with a mixture of digoxigenin-labled *hb* and *lacZ* antisense RNA probes; they are oriented with anterior to the left and dorsal up.

(A) *hb* expression in a +/*P[Kr-lacZ]* embryo that contains two normal copies of the hb^{+} gene, as indicated by the expression of the *Kr-lacZ* reporter gene in central regions (long arrow). Normal patterns of *hb* expression can be detected in both anterior and posterior regions of the embryo.

(B) *hb* expression in an embryo that contains only one normal copy of the hb^{+} gene, as indicated by the absence of *lacZ* expression (small arrow) in the *Kr* domain. *hb* transcripts are detected in both anterior and posterior regions. However, there is an ~2-fold reduction in the levels of *hb* transcripts (compare with A) since the hb^{14} null mutation used in this analysis does not encode a stable transcript (Lehmann and Nüsslein-Volhard, 1987).

(C) Embryo of genotype +/*P[Kr-lacZ]* that was derived from a $TAF_{II}60^{YY}$/+ heterozygous female. The reduction in the maternal dose of the wild-type $TAF_{II}60$ product does not alter the limits or levels of the *hb* expression pattern (compare with A).

(D) Embryo of genotype hb^{14}/+ that was derived from a $TAF_{II}60^{YY}$/+ heterozygous female. The simultaneous reduction in *hb*+ and $TAF_{II}60^{+}$ gene does not alter the *hb* expression pattern beyond the 2-fold reduction in staining due to the hb^{14} null mutation (compare with A and B).